Artykuł przeglądowy *Review*

Young pigeon disease syndrome

JEAN PIERRE DUCHATEL, PIOTR SZELESZCZUK*

Department of Infectious Diseases, Veterinary Faculty, University of Liège, Bd de Colonster, 20 B43, 4000 Liège, Belgium
*Division of Avian Diseases, Department of Pathology and Veterinary Diagnostics, Faculty of Veterinary Medicine, Warsaw University of Life Sciences, 159 Nowoursynowska Str., 02-776 Warsaw, Poland

Duchatel J. P., Szeleszczuk P.

Young pigeon disease syndrome

Summary

The young pigeon disease syndrome (YPDS) is a multifactorial disease in which the pigeon circovirus (PiCV) plays an important role in inducing immunosuppression in infected birds. The PiCV is small and has a non--enveloped icosahedral structure. The YPDS occurs in young birds, usually after weaning at the age of 7-15 weeks, and is characterized by clinical signs such as anorexia, depression, the crop filled with clear yellow liquid, vomiting, diarrhea, polyuria, and ruffled feathers. Macroscopic examination of affected pigeons generally shows atrophy of the bursa of Fabricius (BF) and the thymus. Histopathological examination reveals lesions of acute necrotizing bursitis, characterized by the infiltration of heterophils into the lumen of the follicles and the medulla. Like all tests, PiCV multiplication in cell cultures and the inoculation of embryonated eggs are unsuccessful. The presence of the PiCV is therefore usually established by detecting viral DNA by PCR. There is no specific treatment or vaccine against PiCV infection. When cases of the young pigeon disease are found, secondary infections, as well as bacterial and parasitic diseases, are diagnosed and treated. Further studies are needed to provide answers to many questions regarding this syndrome. All data collected will enable a better management of infections and associated symptoms.

Keywords: pigeons, circovirus, immunosuppression

The young pigeon disease syndrome (YPDS) is a multifactorial disease in which the pigeon circovirus (PiCV) plays an important role in inducing immunosuppression in infected birds. Some authors also describe the disease as „young pigeon sickness". The YPDS occurs in young birds, usually after weaning at the age of 7-15 weeks, and is characterized by clinical signs such as anorexia, depression, the crop filled with clear yellow liquid, vomiting, diarrhoea, polyuria, and ruffled feathers. Generally, 20% of young pigeons are affected, and the mortality rate is about 20%. The clinical picture is strongly influenced by the health status of the flock, i.e., secondary viral infections, as well as bacterial and parasitic diseases. Concurrent infections with *Escherichia coli* are often diagnosed (19).

Pigeon Circovirus

The pigeon circovirus was identified first in the United States in 1993 (36), but a retrospective study showed the presence of similar infections between 1986 and 1993 in the United States, Canada and Australia (37). The virus was subsequently reported in Ireland (23), England (11), Germany (24) and Belgium in 1997 (6). The first clinical cases of PiCV infection in Poland were observed in the late 1990s (27). Wieliczko et al. (35), using PCR, confirmed the presence of the PiCV DNA in 72.2% of tissues of carrier pigeons and in 44.7% of city pigeons from Wroclaw.

Currently, PiCV infection has become cosmopolitan and affects mainly young pigeons (1, 3, 6, 11, 22-26, 28, 29, 36, 37). Most young pigeons are subclinically infected, which is easily explained by the practice of the pigeon sport, which promotes the spread of the virus. Studies have shown the presence of viral DNA in many adult pigeons, especially in the respiratory organs, spleen, kidneys and liver (7, 8).

The PiCV is small (15 to 20 nm in diameter) and has a non-enveloped icosahedral structure. The genome of this virus consists of a single-stranded circular DNA molecule of about 2000 bases, which is unique among viruses that infect animals (30).

Circoviruses possess an ambisense genome organization encoding two major proteins: the replication--associated protein from the virus sense strand (open reading frame (ORF-1)) and the capsid protein (ORF-2) from the complementary sense strand (12, 15, 17, 30, 33). Genome analysis of 11 PiCV from ornamental and meat pigeons of different geographical origins (USA, Europe, China and Australia) and a PiCV from an

Australian turtle with feather malformations (18) showed a similar organization of genomes, ranging in size from 2032 to 2040 nucleotides. The comparison of nucleotide sequences showed an identity ranging from 85.1 to 97.8% (32). Circoviruses because of their small size are highly dependent on cellular enzymes for replication. During the phase of cellular mitosis, viral DNA enters the interior of the nucleus, where it replicates, particularly in tissues with rapid cell division, such as lymphoid tissues and the epithelium of intestinal crypts. The viruses replicate by the rolling circle mechanism (33).

Circoviruses are particularly resistant and stable. The extensively studied porcine circovirus exhibits resistance to chloroform and acid solutions of pH 3, and remains stable at 56 and 70°C for 15 minutes (2). Significant reductions in viral titer are obtained in the presence of potassium monopersulfate, disinfectants based on quaternary ammonium associated with one or three aldehydes, sodium hypochlorite and sodium hydroxide (16).

Pathology

Macroscopic examination of affected young pigeons generally shows atrophy of the bursa of Fabricius (BF) and the thymus (1, 6, 15).

Histopathological examination reveals lesions of acute necrotizing bursitis, characterized by the infiltration of heterophils into the lumen of the follicles and the medulla. Many cells of lymphoid follicles contain large aggregates of basophilic intracytoplasmic inclusions of up to 15 microns in diameter. These inclusion bodies are rarely seen in the spleen, in the thymus, and in the gut- and bronchial-associated lymphoid tissues. Lymphoid depletion and numerous cysts may also be observed in the BF.

Electron microscopy examination of inclusion bodies shows the presence of non-enveloped icosahedral viral particles of 15 to 19 nm in size, arranged in paracrystallin arrays.

The PiCV, like other avian circoviruses, appears to be associated with immune disorders, and this hypothesis is supported by clinical and histological data (13, 20, 25, 29, 30, 36, 37). In young pigeons infected with the PiCV, vaccination against paramyxovirus is often unsuccessful (37). To ensure effective protection against the avian paramyxovirus type 1, it is sometimes necessary in primo-vaccination to inject pigeons twice at an interval of 3 to 4 weeks.

The virus is transmitted horizontally and vertically. Significant numbers of these viruses are found in droppings, and the transmission occurs through ingestion or inhalation of dust contaminated with virulent feces (10, 36). Studies show that most young pigeons are infected horizontally in the loft in the post-weaning period (8).

The PiCV DNA was detected in the organs of embryos and forty-four fertilized eggs from three different lofts; circoviral DNA was detected in 11.4% of embryos (7). Moreover, the presence of viral DNA in large quantities (up to 1×10^7 genome copies per ejaculate) has been demonstrated in the semen of pigeons. Vertical transmission of the virus is not negligible and should be taken into consideration (8, 9).

A method for quantification of viral DNA by polymerase chain reaction quantitative, carried out in pigeons with or no symptoms YPDS showed significantly greater amounts of genome copies in certain tissues such as liver and bursa and serum sick pigeon that infected birds which do not show symptoms (9).

The role and pathogenicity of the PiCV remain to be clarified because an experimental infection of young pigeons with the PiCV failed to reproduce the YPDS (21). Stressors such as overcrowding, poor conditions of transport to the site of release, difficult weather conditions during the return flights during the competition, and secondary infections seem to favor the onset of this multifactorial disease.

Diagnosis

Like all other tests, PiCV multiplication in cell cultures and the inoculation of embryonated eggs are unsuccessful. The presence of the PiCV is therefore usually established by detecting viral DNA by polymerase chain reaction (PCR). The diagnosis of the YPDS requires three conditions to be met. First, the observation of symptoms and gross lesions compatible with this disease should be underpinned by the demonstration of histological lesions and characteristic intranuclear and/or intracytoplasmic viral inclusions, mainly in the bursa. Second, the presence of viral particles with the characteristic morphology of the circovirus, observed by electron microscopy, can be confirmed histological observations. Finally, the presence of large amounts of viral DNA, estimated by the quantitative PCR technique, completes the third part of this diagnosis. During autopsy, the bursa and thymus should be examined with particular care. For instance, 84 and 41% of 32 bursa samples examined were found positive by PCR and histology, respectively (31). Recently, an ELISA method using part of the capsid protein produced in *Escherichia coli* to estimate the antibody titer has been described (4).

Treatment and vaccination

There is no specific treatment or vaccine against PiCV infection. When cases of the young pigeon disease are found, secondary infections, as well as bacterial and parasitic diseases, are diagnosed and treated.

Prevention depends on controlling the factors that favor the onset of the disease in young pigeons infected with the PiCV. Hygiene and sanitary conditions are very important and should be subject to regular monitoring. Since stress seems to play a substantial role, it is important to avoid disturbing the pigeons. This is why young squabs should be weaned as late as

possible, at the age of 24 days, and pigeons of different ages should be kept in separate lofts. It is also important to avoid overcrowding the lofts. Pigeons should be trained in the company of other pigeons from the same loft, but not before they reach the age of 14 weeks. Later, the pigeons can be trained with all other pigeons. The distances and rhythm of training should be carefully considered, taking into account weather conditions and the physiology of birds. Currently, there is no vaccine against PiCV infection. Such a vaccine is likely to be developed by recombinant DNA technology since the PiCV cannot be propagated in cell culture, and inactivated vaccines prepared from virulent viruses from contaminated organs do not always meet the necessary safety requirements. The circovirus is particularly difficult to inactivate completely, and tests to verify the absence of infectious particles in such vaccines are not available.

In a recently published study, the effect of Colombovac Paratyphus® vaccination against paratyphus during the course of infection in young pigeons naturally infected with the pigeon circovirus was examined (5). This vaccination against paratyphus usually has a slight negative effect on the pigeon's fitness. Forty 6-week-old naturally infected pigeons were randomly assigned to 2 equal groups. Pigeons of one group were vaccinated at 6 and 9 weeks of age, and pigeons of the other group were kept as controls. Three weeks after the second vaccination, pigeons were euthanized. No clinical signs were observed in any of the pigeons during the experimental period. There was no statistically significant difference between vaccinated and control pigeons in the number of PCR-positive swabs and blood samples collected from live birds, or in samples taken after necropsy. More of characteristic botryoid inclusions were detected in the vaccinated pigeons than in the unvaccinated ones, but the difference was not statistically significant.

It is important to note, however, that the number of birds with general lesions (bursitis, necrosis and calcification) of the bursa was significantly greater in the unvaccinated group. The vaccination with Colombovac Paratyphus® had no effect on the course of circovirus infection, and no symptoms of the disease were noted during the experiment. Instead, a beneficial effect of vaccination was emphasized (5).

Conclusions

Despite the published epidemiological and molecular data, many questions remain. While pigeon circovirus infections have already been observed for several years, it is still unclear what factors lead to the emergence of the YPDS. What factors promote frequent outbreaks of the YPDS in some lofts while other lofts remain relatively unaffected by the disease? What is the exact pathogenicity of the PiCV? Will a future vaccine against PiCV infection be able to prevent symptoms of the disease? Further studies are needed to provide answers to these questions. All data collected will enable a better management of infection and associated symptoms.

References

1. *Abadie J., Nguyen F., Groizelau C., Amenna N., Fernandez B., Guereaud C., Guigand L., Robart P., Lefebvre B., Wyers M.*: Pigeon circovirus infection: pathological observations and suggested pathogenesis. Avian Pathol. 2001, 30, 149-158.
2. *Allan G., Phenix K., Todd D., McNulty M.*: Some biological and physicochemical properties of porcine circovirus. Zntbl. Vet. Med. B 1994, 41, 17-26.
3. *Coletti M., Franciosini M., Astrubali G., Passamonti F.*: Atrophy of the primary lymphoid organs of meat pigeons in Italy associated with circovirus--like particles in the bursa of Fabricius. Avian Dis. 2000, 44, 454-459.
4. *Daum I., Finsterbusch T., Härtle S., Göbel T., Mankertz A., Korbel R., Grund C.*: Cloning and expression of a truncated pigeon circovirus capsid protein suitable for antibody detection in infected pigeons. Avian Pathol. 2009, 38, 135-141.
5. *Duchatel J. P., Jauniaux T., Smyth J., Habsch I., De Bournonville M., Losson B., Todd D.*: Effect of a commercial Paratyphus vaccine on the development of Pigeon circovirus infection in young pigeons. J. Avian Med. Surg. 2010, 24, 107-114.
6. *Duchatel J. P., Jauniaux T., Vandersanden F., Charlier G., Coignoul F., Vindevogel H.*: Première mise en évidence en Belgique de particules ressemblant à des circovirus chez le pigeon voyageur. Ann. Med. Vet. 1998, 142, 425-459.
7. *Duchatel J. P., Todd D., Curry A., Smyth J., Bustin H., Vindevogel H.*: New data on the transmission of pigeon circovirus. Vet. Rec. 2005, 157, 413-415.
8. *Duchatel J. P., Todd D., Smyth J., Bustin J., Vindevogel H.*: Observations on detection, excretion and transmission of pigeon circovirus in adult, young and embryonic pigeons. Avian Pathol. 2006, 35, 30-34.
9. *Duchatel J. P., Todd D., Willeman C., Losson B.*: Quantification of pigeon circovirus in serum, blood, semen and different tissues of naturally infected pigeons using a real-time polymerase chain reaction. Avian Pathol. 2009, 38, 143-148.
10. *Gerdes G.*: Two very small viruses – a presumptive identification. J. S. Afric. Vet. Assoc. 1993, 64, 2.
11. *Gough R., Drury S.*: Circovirus-like particles in the bursae of young racing pigeons. Vet. Rec. 1996, 138, 167.
12. *Johne R., Fernandez-De-Luco D., Hofle U., Muller H.*: Genome of a novel circovirus of starlings, amplified by multiply primed rolling-circle amplification. J. Gen. Virol. 2006, 87, 1189-1195.
13. *Latimer K., Rakich P., Niagro F., Ritchie B., Steffens W., Campagnoli R., Pesti D., Lukert P.*: An update review of psittacine beak and feather disease. J. Assoc. Avian Vet. 1991, 5, 211-220.
14. *Lefebvre B., Stosskopf O.*: Circovirose dans un élevage de pigeons de chair. Point Vet. 2001, 221, 64-67.
15. *Mankertz A., Hattermann K., Ethers B., Soike D.*: Cloning and sequencing of Columbid circovirus (CoCV), a new circovirus from pigeons. Arch. Virol. 2000, 145, 2469-2479.
16. *Martin H., Le Potier M., Maris P.*: Virucidal efficacy of nine commercial disinfectants against porcine circovirus type 2. Vet. J. 2008, 177, 388-393.
17. *Niagro F., Forsthoefel A., Lawther R., Kamalanathan L., Ritchie B., Latimer K., Lukert P.*: Beak and feather disease virus and porcine circovirus genomes: intermediates between the geminiviruses and plant circoviruses. Arch. Virol. 1998, 143, 1723-1744.
18. *Raidal S., Riddoch P.*: A feather disease in Senegal doves (Streptopelia senegalensis) morphology similar to psittacine beak and feather disease. Avian Pathol. 1997, 26, 829-836.
19. *Raue R., Schmidt V., Freick M., Reinhardt B., Johne R., Kamphausen L., Kaleta E., Muller H., Krautwald-Junghanns M. E.*: A disease complex associated with pigeon circovirus infection, young pigeon disease syndrome. Avian Pathol. 2005, 34, 418-425.
20. *Ritchie B., Niagro F., Lukert P., Steffens W., Latimer K.*: Characterization of a new virus from cockatoos with psittacine beak and feather disease. Virology 1989, 171, 83-88.
21. *Schmidt V., Schlomer J., Luken C., Johne R., Biere B., Muller H., Krautwald--Junghanns M.-E.*: Experimental infection of domestic pigeons with pigeon circovirus. Avian Dis. 2008, 52, 380-386.
22. *Shivaprasad H., Chin R., Jeffrey J., Latimer K., Nordhausen R., Niagro F., Campagnoli R.*: Particles resembling circovirus in the bursa of Fabricius. Avian Dis. 1994, 38, 635-641.
23. *Smyth J., Carroll B.*: Circovirus infection in European racing pigeons. Vet. Rec. 1995, 136, 173-174.

24. *Soike D.*: Circovirus infection bei Tauben. Tierärztl. Prax. 1997, 25, 52-54.
25. *Soike D., Kohler B., Albrecht K.*: A circovirus-like infection in geese related to a runting syndrome. Avian Pathol. 1999, 28, 199-202.
26. *Stenzel T., Piasecki T.*: Zakażenia cirkowirusowe gołębi – główna przyczyna trudnych w terapii zespołów chorobowych. Mag. Wet. 2009, 19, 451-454.
27. *Szeleszczuk P.*: Cirkowiroza czyli zespół nabytego braku odporności (AIDS) gołębi. Hodowca Gołębi Pocztowych 2000, 74, 1-2.
28. *Taras L., Kubicek O., Juranova R., Jurajda V.*: The first demonstration of pigeon circovirus in the Czech Republic based on histology and nested PCR. Acta Vet. Brno 2003, 72, 577-582.
29. *Tavernier P., De Herdt P., Thoonen H., Ducatelle R.*: Prevalence and pathogenic significance of circovirus-like infections in racing pigeons (Columba livia), Vlaam. Diergeneeskd. Tijdschr. 2000, 69, 338-341.
30. *Todd D.*: Circoviruses: Immunosuppressive threats to avian species: A review. Avian Pathol. 2000, 29, 373-394.
31. *Todd D., Duchatel J. P., Weston J., Ball N., Borghmans B., Moffett D., Smyth J.*: Evaluation of polymerase chain reaction and dot blot hybridisation tests in the diagnosis of pigeon circovirus infections. Vet. Microbiol. 2002, 89, 1-16.
32. *Todd D., Fringuelli E., Scott A., Borghmans B., Duchatel J. P., Shivaprasad H., Raidal S., Abadie J., Franciosini M., Smyth J.*: Sequence comparison of pigeon circoviruses. Res. Vet. Sci. 2008, 84, 311-319.
33. *Todd D., McNulty M., Adair B., Allan G.*: Animal circoviruses. Adv. Virus Res. 2001, 57, 1-70.
34. *Twentyman C., Alley M., Meers J., Cooke M., Duignan P.*: Circovirus-like infection in a southern black-backed gull (Larus dominicanus). Avian Pathol. 1999, 28, 513-516.
35. *Wieliczko A., Piasecki T., Houszka M.*: Zakażenia cirkowirusowe gołębi. Medycyna Wet. 2005, 61, 94-97.
36. *Woods L., Latimer K., Barr B., Niagro F., Campagnoli R., Nordhausen R., Castro A.*: Circovirus-like infection in a pigeon. J. Vet. Diagn. Invest. 1993, 5, 609-612.
37. *Woods L., Latimer K., Niagro F., Ridell C., Crowley A., Anderson M., Daft B., Moore J., Campagnoli R., Nordhausen R.*: A retrospective study of circovirus infection in pigeons: nine cases (1986-1993). J. Vet. Diagn. Invest. 1994, 6, 156-164.

Corresponding author: prof. dr hab. Piotr Szeleszczuk, ul. Ciszewskiego 8, 02-786 Warszawa; e-mail: Piotr_Szeleszczuk@sggw.pl